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DR. J. HAMILTON CRAWFORD, Brooklyn, *Editor*

DR. CHARLES A. POINDEXTER, New York, *Associate Editor*

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SUDDEN FALL IN ARTERIAL PRESSURE AS A PRECIPITATING FACTOR IN ACUTE CORONARY THROMBOSIS AND MYOCARDIAL INFARCTION

References to the part that sudden lowering of arterial pressure might play in precipitating coronary thrombosis and myocardial infarction have appeared in the literature during the past few years. Bean, in an analysis of possible causative factors in a group of over 200 cases of myocardial infarction, found three in which collapse, with attendant fall in blood pressure, seemed to be the agent responsible for fresh infarcts. One was in a patient who had had severe hemorrhage from a peptic ulcer. Another had a severe epistaxis. The third collapsed following decompression of the bladder for prostatic retention. All three hearts showed coronary sclerosis. McLaughlin reported a fatal case of peptic ulcer hemorrhage in which the clinical severity of the patient's condition, as well as death, could not be satisfactorily accounted for by the hemorrhage alone. At autopsy, marked coronary atherosclerosis with severe narrowing of the lumina and acute myocardial infarction, were found. No thrombi or actual occlusions were present. There had been no symptoms of cardiac disease. Blood pressure had previously been slightly elevated (around 150). Following the hemorrhage the pressure level fell to 80/60 and remained there. Master, Dack and Jaffee, in a study of coronary occlusion and myocardial infarction as a post-operative complication, found that 60% had fall in blood pressure. They suggest that thrombosis may be caused by changes in volume, speed of flow and pressure within the coronary arteries. Also, that diminution in coronary blood flow may be severe enough to result in infarction without occlusion. A few years before this study, the writer, in collaboration with Saphir, Hamburger and Katz, noted the occurrence of acute coronary thrombosis and myocardial infarction as a post-operative complication in patients with severe coronary sclerosis. It was thought that lowering of arterial pressure was an important factor in precipitating these accidents. In another paper dealing with the anatomic changes in angina pectoris, coronary insufficiency and coronary occlusion, Master and Dack conclude that coronary insufficiency (myomylacia without frank occlusion) may occur when something happens which lowers blood pressure. Bean suggests that lowering of blood pressure by the persistent use of nitrites to relieve acute precordial pain may precipitate coronary thrombosis or myocardial infarction.

It is recognized that other factors in collapse, shock or severe hemorrhage may play a part in precipitating thrombosis or infarction. These may be changes in physical and chemical properties of the blood, diminished oxygen carrying power, (reduced hemoglobin and erythrocyte count) increased oxygen need, tachycardia. However, a common factor in a large percent is a sharp fall in blood pressure. It seems that if the lowered level persists the chances for thrombosis or infarction are increased.

Coronary atherosclerosis, sometimes unsuspected, was present in all but two of the reported cases which have come to the writer's attention. It was present in all of the five cases personally observed in which fall in blood pressure occurred from one to four days prior to symptoms and signs of acute myocardial infarction.

The following cases serve to illustrate the point in question.

Female, Age 75

Present Illness — All complaints referable to the G. I. tract. Carcinoma of pylorus. No cardiac symptoms.

Heart Examination—Generalized enlargement. Faint tones. Systolic murmur loudest at base. Blood pressure 144/88.

Course — On tenth day in hospital, passed a large black fluid stool with free blood. Blood pressure fell to 100/60. Shortly thereafter, severe epigastric pain, weak rapid pulse and pulmonary edema developed. Death six hours later.

Autopsy — Heart, 395 grams. Diffuse myocardial fibrosis. Complete obliteration of left circumflex branch by calcified plaque. Fresh thrombus in right coronary artery. Fresh infarct of lateral wall of left ventricle near the base.

Male, Age 48

Present Illness — Sudden onset of copious tarry stools. R. B. C. 2,830,000. Hemoglobin 40% eight hours later.

Diagnosis — Hemorrhage from peptic ulcer.

Heart Symptoms — Mild substernal pain and some breathlessness on ordinary exertion for two years.

Heart Examination — (a) (2 years before present illness.) Some enlargement to left. Blood pressure 140/88. E. C. G. normal except for left axis deviation.

(b) (5 months before present illness.) Some further increase in size of left heart. Blood pressure 170/100. Blood pressure range from then to time of admission 134-154/90-95. E. C. G., slight depression of RS-T_1, notching of T_1F, left axis deviation.

(c) At time of admission blood pressure 90/65. Pulse rapid, fair quality. Tones distant. E. C. G., no change compared with the one made 5 months before, except lower amplitude of QRS in all lead.

Course — Blood pressure level did not exceed 104/65. On fourth post-admission day, sudden severe persistent precordial pain accompanied by cold sweat and syncope. E. C. G. a few hours later showed small slurred QRS complexes, definite Q_3, slightly inverted T_1, slightly elevated RS-T_3, almost absent R_3F, upright T_3F. Fever and leucocytosis developed. Diagnosis: Acute myocardial infarction. On the eighth, eleventh, and fourteenth days, abrupt rise in temperature to approximately 101.5, each time associated with dyspnoea, sweating, increase in leucocyte count but no precordial pain. Temperature returned to normal within 24 hours each time. Serial E. C. Gs. showed change from T_3 to T_2 type between the eighth and eleventh day with further decrease in amplitude of QRS. T_3F did not change. Blood pressure not over 104/65 until after the eleventh day. Discharge eight weeks after admission. Blood pressure level then averaged 140/70. E. C. G. three months after first cardiac insult was of Q_3-T_3 type with very small QRS complexes in standard and precordial leads plus inverted T_3F which had not been present previously. One month later the E. C. G. was the same. Seven months after first infarction, severe persistent precordial pain at 3:30 a.m. Death at 1:10 p.m. E. C. G. at 12:45 showed practically absent QRS in all leads, depressed RS-T_2, elevated RS-T_3F, markedly depressed RS-T_3F.

Diagnosis — Acute myocardial insufficiency due either to fresh thrombus of remaining unoccluded branch or fresh infarction or both.

Autopsy — Heart enlarged. Marked narrowing of right coronary ostium by atheromatous plaques. Similar condition of left but less. Right coronary artery completely occluded by old (organized) thrombus extending from the ostium almost to posterior descending branch, involving also the Ramus Marginis Acuti well past its first bifurcation. Lumen of posterior descending branch narrowed by atheromatous plaques. Lumina of anterior descending and circumflex branches of left coronary markedly narrowed by tubular plaques extending well into smallest sub branches of each. Old, healed infarct in anterior wall of left ventricle. Both old and very recent infarction of the posterior wall of the left ventricle and the posterior papillary muscle. The fresh infarct was in the area supplied by the terminal branches of the left circumflex and right coronary arteries. In each of these, just proximal to the anastomotic branching, was a very recent thrombus.

In both of these cases the sequence of hemorrhage from the gastro-intestinal tract, sudden fall in blood pressure, then later, coronary thrombosis and/or myocardial infarction seems too clear-cut to be disregarded. In both, the previous blood pressure level was known. Both had been under conditions of daily observation for some time. Because of the absence of cardiac symptoms in the first case, it does not seem logical to assume that the infarction occurred during the ten days prior to epigastric pain and pulmonary edema. The age of the fresh thrombus and infarct was not compatible with their having occurred prior to admission to the hospital. This patient was not dehydrated.

In the second case the fall in blood pressure was not only sudden but the low level persisted. From the anatomic picture and electrocardiographic changes, the following sequence of events may be postulated: (1) Infarction of anterior wall of the left ventricle (without thrombosis), four days after hemorrhage due to peptic ulcer because of sclerotic obliteration of the descending branch in the presence of marked narrowing of the circumflex branch plus diminished volume flow of blood. (2) Sometime during the succeeding seven to fifteen days, possibly while the blood pressure level was still low, thrombus formation started in the right coronary artery and gradually increased in extent with resulting infarction of the posterior wall of the left ventricle. This was a very large infarct and possibly attained its ultimate size over a period of time or represented the "building up" of several smaller acute infarctions as suggested by the series of clinical episodes following the first attack of precordial pain. (3) For five months after leaving the hospital he lived a relatively normal life, then died after ten hours of severe pain. Fresh thrombi in the terminal branches of the right coronary and left circumflex branch with further infarction of the posterior wall of the left ventricle occurred as the terminal event.

In view of the clinical history and the electrocardiogram made shortly after the hemorrhage which did not show significant changes compared with previous ones, it does not seem likely that either of the old infarcts could have occurred prior to the hemorrhage. The possible role of the anemia itself in precipitating the first infarctions and thrombus must be considered, but it was not severe and prompt and frequent transfusions had brought the blood picture almost to normal before the attack of precordial pain believed to be evidence of the first infarction. Hence the abrupt fall in blood pressure and persistence of a low level appears to be the significant factor in precipitating the first infarct. Dehydration was not evident. Unfortunately, studies of volume, velocity of blood flow and blood viscosity were not made.

The occurrence of the final thrombosis and infarction in this case during a period of normally lowered blood pressure (sleep) raises the question as to whether such lower levels of pressure could account for part, at least, of the "coronary accidents" occurring during sleep. The actual amount of fall in blood pressure necessary may be relatively slight. As Saphir has pointed out, the final causes for a temporary coronary or myocardial insufficiency, may appear insignificant. From personal observation and interpretation of the experience of others, suddenness of the fall appears more important than extent.

The possible role of a fall in blood pressure in the causation of coronary thrombosis and myocardial infarction, is based on clinical observation. In view of the reduced arterial channels in coronary sclerosis it seems mechanically possible. It has appeared of sufficient importance to several observers to warrant urging the most careful and complete cardiovascular study of prospective surgical patients of an age likely to have coronary sclerosis. Especial emphasis is placed on paying attention to the blood pressure during and immediately after operation. There is also clinical evidence to warrant equal concern over fall in blood pressure associated with any extra-cardiac accident such as hemorrhage and shock.

Walter S. Priest, M. D.
Chicago, Illinois